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Ecology of Zoonotic Infectious Diseases in Bats: Current Knowledge and Future Directions

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Impacts

- Bats are hosts to a range of pathogens, some of which are known to infect and cause disease in humans and domestic animals. Human activities that increase exposure to bats will likely increase the opportunity for these infections to spill over from bats to humans in the future.
- Understanding the impacts of anthropogenic changes on infection dynamics within bat populations is necessary to predict and prevent human infections of bat origin. However, this initially requires understanding both bat populations and the dynamics of infections within them.
- We propose that a combination of field and laboratory studies are needed to create data-driven mathematical models to elucidate aspects of bat ecology that are most critical to the dynamics of emerging bat viruses.

Keywords:
Epidemiology; zoonoses; mathematical modeling; Nipah virus; public health; rabies; filovirus; coronavirus

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Summary

Bats are hosts to a range of zoonotic and potentially zoonotic pathogens. Human activities that increase exposure to bats will likely increase the opportunity for infections to spill over in the future. Ecological drivers of pathogen spillover and emergence in novel hosts, including humans, involve a complex mixture of processes, and understanding these complexities may aid in predicting spillover. In particular, only once the pathogen and host ecologies are known can the impacts of anthropogenic changes be fully appreciated. Cross-disciplinary approaches are required to understand how host and pathogen ecology interact. Bats differ from other sylvatic disease reservoirs because of their unique and diverse lifestyles, including their ability to fly, often highly gregarious social structures, long lifespans and low fecundity rates. We highlight how these traits may affect infection dynamics and how both host and pathogen traits may interact to affect infection dynamics. We identify key questions relating to the ecology of infectious diseases in bats and propose that a combination of field and laboratory studies are needed to create data-driven mechanistic models to elucidate those aspects of bat ecology that are most critical to the dynamics of emerging bat viruses. If commonalities can be found, then predicting the dynamics of newly emerging diseases may be possible. This modelling approach will be particularly important in scenarios when population surveillance data are unavailable and when it is unclear which aspects of host ecology are driving infection dynamics.

Introduction

Emerging infectious diseases in wildlife threaten global biodiversity and public health (Daszak et al., 2000). Bats can host a range of zoonotic and potentially zoonotic pathogens. In addition to rabies (RABV) and other lyssaviruses (e.g. Kuzmin et al., 2008b; Streicker et al., 2010), bats have been identified as the likely reservoir for severe acute respiratory syndrome (SARS) coronavirus (CoV) (Li et al., 2005; Cheng et al., 2007; Vijaykrishna et al., 2007), Hendra (HeV) (Halpin et al., 2000), Nipah (NiV) (Chua et al., 2002; Hsu et al., 2004; Reynes et al., 2005), Ebola (EBOV) (Leroy et al., 2005) and Marburg (MARV) viruses (Monath, 1999; Peterson et al., 2004a,b; Towner et al., 2007). Most recently, a new distinct lineage of influenza A virus has been discovered in little yellow-shouldered bats (*Sturnira lilium*, family Phyllostomidae) in the Americas (Tong et al., 2012) and a range of paramyxoviruses in bats from four continents (Drexler et al., 2012). Given the potentially devastating effects of these emerging diseases on public health and wildlife conservation (e.g. EBOV and gorillas; Bermejo et al., 2006), it is crucial that we improve our understanding of how bat ecology may influence disease dynamics and their propensity to serve as reservoirs for emerging pathogens (Messenger et al., 2003; Calisher et al., 2006; Wong et al., 2007).

Ecological drivers of pathogen spillover and emergence in novel hosts, including humans, can be a complex mixture of processes (Lloyd-Smith et al., 2005, 2009). Clearly, human activities that increase exposure to bats will increase the opportunity for infections to be transmitted between bats and humans or to intermediate hosts such as pets and livestock. However, our knowledge of how and why emerging pathogens spill over from bats is limited, and improved understanding of these processes will require cross-disciplinary approaches. Traits of the pathogen and the human–pathogen interactions at the cellular level, such as evolutionary mutation rates and receptor-binding affinity, are important when trying to understand spillover and emergence (Moya et al., 2004; Pulliam, 2008; Pulliam and Dushoff, 2009). However, these traits are proximate causes, and the ultimate drivers of spillover and emergence are ecological (Lloyd-Smith et al., 2009). Therefore, understanding host ecology and elements of the human–animal interface are essential in the context of pathogen spillover events. For example, host population structure and seasonality may affect the dynamics, viral diversity and virulence of infection in the host population, which may in turn affect the risk of spillover (Boots et al., 2004; Turmelle and Olival, 2009). Attempts to understand how host ecology impacts the dynamics of infection are relatively few in wild animal populations and fewer still for bat populations, and attempts at understanding how infections themselves evolve to persist in wildlife hosts with different ecologies are rarer still. To understand the role of host ecology in disease dynamics, we recommend combining field and experimental methods iteratively to parameterize mechanistic models, as well as integrative modelling in a comparative context, between species, population cohorts and pathogens (Plowright et al., 2008b; Plowright et al., 2011; George et al., 2011); important also in this context is the use of models to prioritize and plan field studies (Restif et al., in press). Only once host and pathogen ecologies are united, can reliable predictions be made regarding ecological drivers of spatiotemporal infection dynamics and spillover (Keeling and Rohani, 2008).

Infection dynamics in bats are likely influenced by the unique ecology of this diverse group of mammals. There are several ways in which bats differ from other similarly sized mammals, including potential for rapid and widespread dispersal, highly gregarious social structures, long lifespans and high survival, with low fecundity (Calisher et al., 2006). The majority of the studies on bat infections to date have focused on bats and zoonotic viral infections, but the ecological generalities may be similar among bacterial (Kosoy et al., 2010; Bai et al., 2011) and fungal infections of bats (e.g. *Histoplasma capsulatum*, *Geomyces destructans*) (Taylor et al., 2005; Blehert et al., 2009; Frick et al., 2010; Foley et al., 2011; Puechmaille et al., 2011). Although widely recognized, few studies have involved explicit hypothesis testing regarding pathogen associations and the unique ecological characteristics of bats.

In this article, we review how interactions between the ecologies of the host(s) and pathogen drive the infection dynamics within host populations and highlight the importance of considering both when investigating spillover events and dynamics. However, there is currently a paucity of studies on the ecology of bat infectious diseases, sometimes leading us to reference some studies in multiple contexts. Therefore, we end each section with a summary of the key unanswered questions relating to the ecology of bats and their infections, then make suggestions as to how future research could address these questions.

Host Ecological Strategies Driving Bat Infection Dynamics

Many aspects of wildlife biology are strongly seasonal, and the ecological strategies that hosts use for coping with seasonal variability may influence disease transmission (Altizer et al., 2006) and emergence. Seasonal host dynamics may be coupled to disease dynamics in, and emergence from, bats by influencing contact rates and susceptibility of the population to infection. Major mechanisms for coping with seasonality in temperate zone bats include restricted birthing periods, migration, use of coloniality and torpor. Each of these strategies may affect population density, contact rates and immune response, thus leading to spatiotemporal variation in infection dynamics. Prevalence of rabies viruses (RABV), CoV and astroviruses (AstV) in bats has been reported to exhibit seasonal dynamics (Mondul et al., 2003; Drexler et al., 2011; Patyk et al., 2012). Changes in the seasonal timing of RABV prevalence among bats in particular appear to correlate with ecological characteristics of the host species (George et al., 2011). However, mechanistic explanations of how host ecological strategies for coping with seasonal variability influence infection dynamics in bats are largely lacking.

Host reproduction and survival as major drivers of bat disease dynamics

Many species of bats in both temperate and tropical zones exhibit highly synchronized parturition (Mutere, 1968; Fleming et al., 1972; Heideman et al., 1992; Bernard and Cumming, 1997; Racey and Entwistle, 2000; Porter and Wilkinson, 2001; Greiner et al., 2011), which can dramatically alter population contact rates and susceptibilities for short time frames. The influx of susceptible young is a crucial driver of infection dynamics (Anderson and May, 1979). However, the role and strength of different host reproduction strategies on disease dynamics are as yet unknown, and only rare examples exist in the literature. Simulations of spatial models of raccoon rabies in the USA, a relatively well-studied system, suggest that increasing seasonality in births as latitude increases leads to increasingly asynchronous rabies dynamics (Duke-Sylvester et al., 2011). Hosseini et al. (2004) were able to demonstrate that seasonal breeding of house finches (*Carpodacus mexicanus*), along with seasonal social aggregation and partial immunity, was key in explaining the specific semi-annual pattern of prevalence seen in *Mycoplasma gallisepticum* conjunctivitis. Many bat species also show marked differences between the sexes in distribution and behaviour during the warmer months, including the use of torpor (discussed below) and degree of coloniality (Cryan et al., 2000; Senior et al., 2005; Weller et al., 2009). Sex differences in behaviour and distribution of bats during times of year when the potential for disease transmission is greatest may also have important implications for disease dynamics, although typically behaviour and population demographic data exist for only a single sex (Weller et al., 2009; O'Shea et al., 2010; Kerth et al., 2011).

Unlike other emerging diseases of bat origin, there is considerable background field and laboratory information on bat RABV; however, even with this relative wealth of information, it was only recently that attempts were made to understand how host ecological strategies influence infection maintenance (Dimitrov and Hallam, 2009; Dimitrov et al., 2008; George et al., 2011). Turmelle et al. (2010a) showed empirically contrasting temporal patterns of RABV exposure in *Tadarida brasiliensis* at different types of roosts during the reproduction season, which suggested increased RABV exposure after parturition in cave colonies. Specifically, cave colonies in this system are known to harbour up to a million individual bats (Betke et al., 2008), mostly reproductively active females, leading to a short window of time where population size doubles following parturition, and contact rates between adult and newborn bats are elevated during early lactation. Individual- and roost-level variation in physiological stress was also demonstrated in this system (Allen et al., 2011), although direct impacts of stress on susceptibility or immune response of bats to RABV or other pathogen infections have not been well characterized to date (but see Smith, 1981).

In modelling RABV in big brown bats (*Eptesicus fuscus*) in Colorado, George et al. (2011) found that interaction between the timing of the annual birth pulse and the RABV incubation period (extending through torpor) was important for RABV perpetuation in the bat population. Host survival rates were important, particularly during hibernation when lower mortality rates facilitated host population persistence, as was the seasonal pulse of susceptible juveniles entering the population in summer (O'Shea et al., 2010, 2011b). Thus, George et al. (2011) were able to show that seasonality in both births and deaths were important factors in RABV persistence *E. fuscus*. Plowright et al. (2011) examined the transmission dynamics of HeV in Australian pteropid bats using a mathematical model parameterized with field and laboratory data. The authors speculated in models of HeV in Australia that it was population connectivity and immunity that played a key role in the infection dynamics, rather than a simple influx of naïve young. It is noteworthy that incorporating waning maternal immunity into models describing HeV–*Pteropus* interactions provided a better fit to the data, therefore demonstrating that not only does the influx of naïve young affect disease dynamics, but that the rate of inflow of susceptible hosts is important in driving pathogen dynamics.

A 3-year study of CoV, AstV and adenoviruses (AdV) in a colony of *Myotis myotis* in Germany demonstrated that RNA viruses (CoV and AstV), but not DNA viruses (AdV), were strongly amplified during colony formation and after parturition and that bat population and virus dynamics were correlated (Drexler et al., 2011). This study also suggested that the breeding success of the colony was not affected by CoV or AstV and that these viruses had seemingly little pathogenic influence on bats, although the lack of individual tracking and follow-up precludes conclusive evidence to support this idea. However, Drexler et al. (2011) concluded that the correlation between bat and virus dynamics suggested that both coloniality and a birth pulse of susceptible hosts may be important for infection dynamics of RNA viruses.

Migration and coloniality as major drivers of bat disease dynamics

Different species of bats manifest distinctive differences in both migratory behaviour (e.g. long distance versus restricted local movements) and population density within colonies (e.g. hundreds per square metre versus solitary or very small groups), yet the impact of such differences in terms of disease dynamics have not been well studied. The majority of bats in temperate zones also cycle between summer maternity roosts and winter hibernacula, and population density, sex and species composition can vary widely between these two types of seasonal roosting aggregations. Indeed, the effects of often very different behaviours between male and female bats (Weller et al., 2009) and the role of increased contact during mating seasons have yet to be explored in relation to infection dynamics. Tropical, non-hibernating bats may use migration and coloniality for other reasons, for example, to track the seasonal availability of fruit and flowers (Thomas, 1983; Richter and Cumming, 2006). As fission–fusion social structures are increasingly being recognized in bats (Storz et al., 2001; Kerth et al., 2011), these may interact with other life history traits to have a profound impact on pathogen transmission rates and persistence.

Seasonal variation in the prevalence of infection is likely impacted by changes in density during migration or colony formation, which affect contact rates and thus disease dynamics (Altizer et al., 2011). Some of the clearest examples of how seasonality and 'coloniality' may affect and drive infection dynamics come from human systems. The episodic dynamics of measles epidemics in the absence of vaccination, for example, driven by school terms (Finkenstadt and Grenfell, 1998; Finkenstadt et al., 1998) or fluctuating agrarian systems (Ferrari et al., 2008; Bharti et al., 2011) suggest changes in contact rates of susceptible hosts may be key in driving infection dynamics. As mentioned, Hosseini et al. (2004) also demonstrated that seasonality in coloniality was important for predicting *Mycoplasma* dynamics in house finches. Although not zoonotic, in bat systems *G. destructans* dynamics and the current epidemic in North America appear to be especially driven by coloniality and migration (Frick et al., 2010). Comparative studies may help to elucidate the impact of variation in seasonal ecologies among or within species affected by the same species or variant of pathogen. For example, the seasonality in aggregation and population dynamics of big brown bats in relation to maintenance of RABV in temperate Colorado (George et al., 2011) could differ in warm temperate and tropical areas where the host species also occurs (Kurta and Baker, 1990; Turmelle et al., 2011).

Assuming a metapopulation model of connectivity, models of HeV predicted that decreased migration of *Pteropus* bats in Australia, possibly because of urbanization and changes in food availability, could influence the intensity and duration of HeV epidemics (Plowright et al., 2011). Decreased migration could give rise to more intense but shorter HeV outbreaks after local viral reintroduction. The mechanism proposed for such an increase in epidemic intensity was that decreased bat migratory behaviour could lead to a decline in transmission between colonies and, therefore, reduced inter-colony exposures and resulting immunity within colonies. This proposed loss of immunity leads to increased epidemic size and more rapid fade-out once infection is re-introduced into a susceptible colony. Thus, reduced migration and population connectivity were suggested as a mechanism that could increase the amplitude of the seasonal outbreaks and increase the probability of spillover. This relationship may be relevant to a variety of bat-associated pathogens, where hosts exist as a metapopulation and humans alter connectivity of populations.

Use of torpor as a major driver of bat disease dynamics and seasonality

Many species of bats are facultative heterotherms capable of using deep torpor during periods of physiological stress to offset energy and water deficits (Humphries et al., 2002; Speakman and Thomas, 2003). For tropical species, reliance on short-term and shallow torpor to offset energy deficits is less well described and may be more prevalent than is currently appreciated (Coburn and Geiser, 1998; Stawski et al., 2009; Stawski and Geiser, 2010). The role of torpor in infection dynamics is largely unstudied (Bouma et al., 2010; Moore et al., 2011). Species of bats that differ in their use of migration also often differ in their use of torpor. Torpor typically reduces pathogen replication rates (Sulkin et al., 1960; Luis and Hudson, 2006) and hence lengthens incubation periods (Bouma et al., 2010) and prevents host contact; these in turn influence the seasonal force of infection (the rate at which susceptible individuals contract infection). Additionally, it likely provides an efficient mechanism for overwintering of some infections.

Multidisciplinary studies, using both experimental data and integrative mathematical models, can be used to determine the importance of temporal changes in torpor use in driving temporal changes in infection prevalence, as was done by George et al. (2011). We argue that the effects of both torpor and changes in contact rates during migration and colony formation might only be differentiated using mechanistic process models. George et al. (2011) found a clear indication for torpor being a key factor in allowing perpetuation of RABV through the hibernation period, through prolonged incubation period and reduced mortality; this enabled RABV infection to persist in the population until susceptible individuals from the annual birth pulse could become infected and continue the cycle. However, RABV is also capable of perpetuating in a variety of bat hosts that are not known to utilize torpor, and even the same host species under varied ecological circumstances (i.e. warmer tropical climates), which begs the question of which factors allow RABV to avoid population fade-out under these circumstances (see Dimitrov et al., 2007, 2008).

An interesting but non-zoonotic example of the importance of torpor on pathogen dynamics is white-nose syndrome (WNS) in bats. The associated fungus *G. destructans* has been detected in 19 US states and two Canadian provinces, and WNS disease has been linked to the deaths of more than five million bats in North America (Blehert et al., 2009; Frick et al., 2010). The causative psychrophilic (cold-loving) fungus colonizes the skin causing devastating lesions by eroding, digesting and replacing living skin tissues with the large surface areas of bat wings being the primary target of infection (Cryan et al., 2010). Hibernation depresses all physiological processes, including immunological processes (Bouma et al., 2010), and thus during deep torpor *G. destructans*, which grows only at temperatures <20°C, is able to replicate more efficiently than would be possible in a bat that has aroused or does not utilize torpor to the same degree. Thus, although *G. destructans* may show the opposite temperature pattern of infection to RNA viruses, it emphasizes the potential importance of torpor on infection dynamics.

- Future directions for research to address the role of host ecological strategies in driving bat infection dynamics

Longitudinal infection and demographic data are required to understand how birth, survival, coloniality, migration and torpor affect infection dynamics within bat populations. Frequently, researchers monitor one part of the process, such as survival (Papadatou et al., 2011), infection (Drexler et al., 2011; Field et al., 2011) or seroprevalence (Breed et al., 2011). However, some studies captured long-term longitudinal data. Hayman et al. (2012a,b) concurrently monitored each in *Eidolon helvum* African fruit bats, and George et al. (2011) and Plowright et al. (2011) synthesized demographic data with infection-related data from field and laboratory studies to give insights into potential mechanisms that drive infection dynamics. Capture–recapture data should be collected to allow estimation of both demographic parameters (O'Shea et al., 2011a,b; Papadatou et al., 2011; Hayman et al., 2012b), while age-specific infection data are particularly useful because they allow such estimation of factors, such as force of infection (Hayman et al., 2012a). Similarly, host population genetic data can be informative to measure connectivity between bat populations, as has been done for NiV and HeV (Olival, 2008). Studies should also attempt to capture the seasonal variation, such as pre-, during- and post-parturition and migration or hibernation seasons, and consider the potential effects of often very different migratory behaviours between male and female bats of some species (Weller et al., 2009). In all cases, however, longitudinal sampling of individuals in experimental and/or captive studies may be required to interpret field-related data. For example, Sulkin et al. (1960) demonstrated that torpor prevents viral replication for the duration of hibernation, and, along with describing antibody titre decays, Rahman et al. (2011) demonstrate that recrudescence of NiV may occur in bats, which may be used to interpret field data. Statistical and mechanistic models, such as susceptible-infected-recovered (SIR)-based models (see Box 1), can then be used on these data to determine which factors best predict infection dynamics. Comparison of mechanistic models, using careful statistical approaches to determine model fitting (Restif et al., in press), may be required to ascertain whether seasonal birth pulses or changes in contact rates, modelled as step functions (e.g. Duke-Sylvester et al., 2011) or seasonal forcing (e.g. Hosseini et al., 2004) for example, drive infection dynamics. Meta-population models may be required to capture all aspects of the system (e.g. Plowright et al., 2011), but must also take into account what is observed in small isolated populations (Peel et al., 2012).

Multispecies, Multipathogen Dynamics

Very few studies in any wildlife system have adequately described either multihost pathogen dynamics or multipathogen dynamics in a single host species. This is hardly surprising given the complexities involved in understanding single pathogen–single host dynamics. However, there are important studies that suggest both multihost and pathogen dynamics are important in other non-bat systems, and empirical data that suggest these situations may

Box 1 Classifying bat populations into susceptible (S), exposed (i.e. incubating infection; E), infectious (I) and recovered (immune; R) classes allows analysis of infection dynamics in bat populations. Three alternative model structures used to model the lyssavirus transmission period for different bat populations and their lyssaviruses are shown. Parameters are as follows: *b*-birth rate; β-transmission coefficient; γ-rate of recovery (seroconverstion); ε-disease-induced mortality; *d*-'natural' mortality; ρ-probability of infection causing disease

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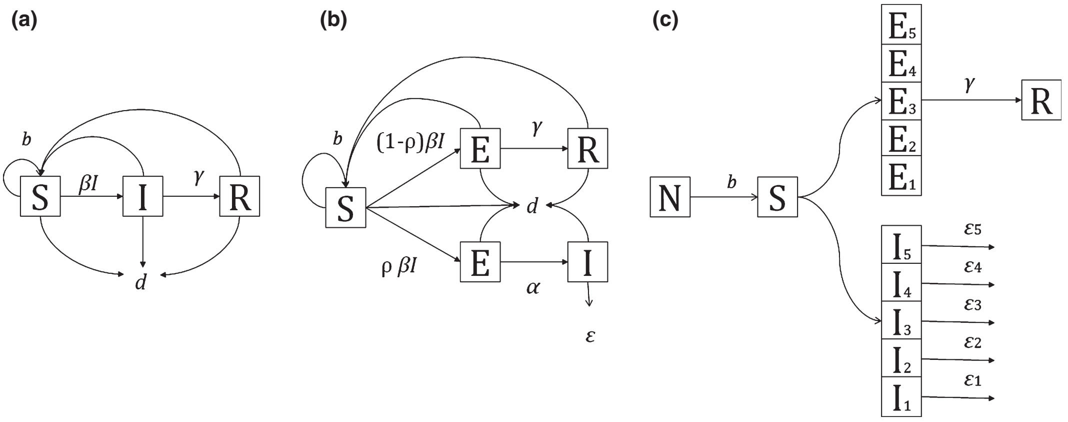
(A) The SIR model structure used by Amengual et al. (2007) to understand possible asymptomatic European bat lyssavirus-1 infection dynamics in *Myotis myotis* bats.

(B) The SEIR model structure used by George et al. (2011) to understand rabies virus (RABV) dynamics and persistence in *Eptesicus fuscus*. Three 'seasons' were modelled: spring birthing/pre-transmission; summer transmission (with this structure) and winter hibernation (with no transmission).

(C) The SEIR structure used by Dimitrov et al. (2008) to understand how different immunotypes of host allowed RABV persistence in *Tadarida brasiliensis* bats and how RABV might select for different immunotypes in populations. N is population size (= S+E+I+R). Differing infectivity and mortality rates, *d*, were modelled, and infection and transmission or recovery was modelled with individual-based models.



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occur in bat infection systems. Through analysis of time series data, Telfer et al. (2010) demonstrated statistically that in a parasite community, including a virus, protozoan and two bacteria, within individual field voles (*Microtus agrestis*), risk of infection was altered by concurrent infection to a greater extent than by age or season. Lello et al. (2004) demonstrated that, in a rabbit (*Oryctolagus cuniculus*) population, gut helminth community parasites either compete or exist in mutualistic relationships. Few studies have considered multiple infections in bats (Drexler et al., 2011). Muhldorfer et al. (2011) detected infections in 12% of 486 bats from 19 European bat species, detecting co-infection with herpesviruses in five bats, but were unable to infer much from this study as it was based on opportunistic sampling. Also, in Thailand, two distinct clades of Nipah virus were found to circulate in the same colony of the fruit bat, *Pteropus lylei* (Wacharapluesadee et al., 2010; Rahman et al., 2010)

From a multihost community perspective, Davies and Pedersen (2008) found that host-relatedness and geographical range overlap were significant predictors of pathogen sharing among primates. Some viruses, notably RABV, are promiscuous, infecting multiple host species. Streicker et al. (2010) demonstrated that although RABV variants predominantly circulate within single host species, they are able to spill over into other species. Similar to Davies and Pedersen (2008), Streicker et al. (2010) documented highly asymmetrical patterns of cross-species RABV transmission in the North American bat fauna, with host-relatedness and geographical range overlap being the strongest predictors of cross-species transmission, thus also suggesting an important influence of host sympatry. Whether these factors affect cross-species transmission of other lyssaviruses within Chiroptera, or from bats to other mammals, is not known, because isolations are often few and serological findings may be due to cross-reactivity between related species or variants, complicating interpretation (e.g. Wright et al., 2010). European bat lyssaviruses 1 and 2 (EBLV-1, EBLV-2) appear to show a very narrow host range in Europe. EBLV-1 circulates in Serotine bats (*Eptesicus serotinus*) and EBLV-2 in Daubenton's bat (*Myotis daubentonii*); this host fidelity is, however, not complete, as the first isolation of EBLV-2 was from a Pond bat (*Myotis dasycneme*), and in Spain, EBLV-1 sequences have been recovered from several bat species (Serra-Cobo et al., 2002; Amengual et al., 2007). The importance of cross-species transmission events in seasonally changing communities may vary with respect to pathogen or variants of a pathogen, but is not really clear for any system. In some cases, infection cycles may be maintained in co-roosting species without cross-species transmission. For example, Kuzmin et al. (2011) discovered that Commerson's leaf-nosed bat (*Hipposideros commersoni*) is a possible reservoir of Shimoni bat virus (SHIBV, a lyssavirus), while Egyptian fruit bats (*Rousettus aegyptiacus*) and *Miniopterus* spp. bats in the same caves were seropositive against Lagos bat virus (LBV) and West Caucasian bat virus (WCBV) (Kuzmin et al., 2008b), thus suggesting that at least for these lyssaviruses, infections may circulate among specific host species and transmission may be minimal among sympatric bats. Moreover, Cui et al. (2007) reported clustering of CoV sequences from geographically separated Vespertilionid bats of the same species, even for co-roosting bats. Coronaviruses from Rhinolophidae bats, however, did not share this feature and appear to have undergone a number of host shifts.

- Future directions for research to address the role of multiple hosts in infection dynamics and multiple pathogens in infection dynamics.

Research programmes that focus on multiparasite and/or multihost systems, following the approach described previously, will help advance our understanding of the ecology of bat diseases. Particular care is needed to consider the markedly reduced statistical power available when considering the dynamics of co-infections, and modelling to help plan empirical data collection would be particularly beneficial for such studies (Restif et al., in press). These studies could particularly benefit from community ecology approaches, testing for inter-specific interactions, such as described by Telfer et al. (2010). Additionally, more detailed molecular techniques and robust co-evolutionary studies could be incorporated that tease out cross-species transmission events (e.g. Cui et al., 2007; Streicker et al., 2010) and infection dynamics (e.g. Drexler et al., 2011).

Host Ecological Strategies as Drivers of Pathogen Virulence

Factors relating to the use of torpor and migration, as well as spatial structure, may select for different optimal levels of virulence in bats, which are long-lived and may acquire immunity to diseases such as RABV (Turmelle et al., 2010b). Intrahost pathogen replication is generally very temperature-dependent, and so seasonal torpor usually suspends it (Sadler and Enright, 1959; Sulkin et al., 1960; Luis and Hudson, 2006). Thus, species and sex differences in torpor behaviour may affect the coevolution of pathogen variants and their transmission rates and generation times (i.e. lineage birth and death processes). Boots et al. (2004) showed theoretically how large shifts in virulence may occur in pathogen populations. They modelled infection in long-lived species that acquire immunity following infection, and as a result of bi-stability in evolutionary dynamics caused by the host's local contact or social population structure, large shifts in virulence could be predicted (Boots et al., 2004). Furthermore, Boots and Sasaki (1999) demonstrated that greater potential for long-distance spread of the pathogen can increase virulence by reducing local selective pressure on the pathogen by exhausting the supply of susceptible hosts from a population. More work, both theoretical and empirical, is required to understand these phenomena in natural host populations. Gandon (2004) used theoretical models to predict the effect of multiple hosts on infection virulence. Interestingly, Gandon's work predicts that if an infection adapts to the most abundant host, then it may well be maladapted to other, less frequent hosts, and this maladaptation may lead to avirulence or hypervirulence in the new or less common host. Thus, decreased interspecies transmission leads to pathogen adaptation to a more abundant host species, whereas increased inter-species transmission leads to more generalist virulence strategies. These theoretical findings may be especially interesting for studies of bats, because of the very large colony sizes that some bats can reach.

Despite these theoretical studies, there is currently little empirical evidence for bat host ecology affecting pathogen virulence. The best example comes from RABV, where up to 30 different lineages of bat RABV may exist in the United States (Messenger et al., 2002; Streicker et al., 2010), and it appears that different bats maintain host-specific RABV variants within populations (Shankar et al., 2005; Streicker et al., 2010). Following the control of canine rabies, the majority of indigenously acquired human rabies infections in the United States are attributable to RABV variants associated with bats (Messenger et al., 2002, 2003). It is unclear whether this mortality from bat rabies is from unrecognized exposure (e.g. humans being bitten during sleep and so not obtaining post-exposure prophylaxis) or is owing to differences in properties of RABV variants; however, some data are suggestive of the latter. Most human rabies deaths in the United States during the past decade are linked to variants associated with the silver-haired bat (*Lasionycteris noctivagans;* almost exclusively this species in the western United States) and *Perimyotis subflavus,* the tricoloured bat (Krebs et al., 2000a,b; Messenger et al., 2003; Franka et al., 2006). In most cases of human rabies associated with bat RABVs, there is no definitive bite history (Rupprecht et al., 2002). The high proportion (74%) of human rabies cases attributable to bat RABV variants cannot be explained by a higher frequency of exposure to these species, as they tend to be non-synanthropic. Two hypotheses have been proposed to account for this phenomenon: 1) the RABV variants associated with *L. noctivagans* and *P. subflavus* are widespread among bat populations and moving through undetected host reservoirs; 2) these variants are more infectious than others. Streicker et al. (2010) provided evidence that neither variant dominates spillover infections into other bat hosts, as indicated by the relatively low proportion of cross-species infections detected with these RABV variants. Concerning the latter hypothesis, laboratory studies suggested higher *in vitro* pathogenicity and neuroinvasiveness of the variant associated with *L. noctivagans* than in a variant isolated from carnivores (Morimoto et al., 1996; Dietzschold et al., 2000; Faber et al., 2004); however, further studies are required because of the uncertain host origin of one of the viruses isolated from a human case (I. Kuzmin, personal communication). However, studies have not adequately investigated comparative pathogenicity *in vivo* in bats relative to the diversity of bat RABV variants that persist naturally (but see Turmelle, 2009).

Lasionycteris noctivagans belongs to a unique group of long-distance latitudinal migrants that roost in trees throughout the year ('tree bats'). Ecological strategies of tree bats differ from other bats in North America and likely drive RABV dynamics in unique ways. Although there were no dominant RABV variants, Streicker et al. (2010) show a propensity for tree bats to asymmetrically infect other bat hosts with RABV. RABV variants associated with tree bats are also among the most recent to emerge (Franka et al., 2006). The winter habits of *L. noctivagans* are poorly known, but intermittent torpor with frequent activity at lower latitudes is likely (Geluso, 2007). Females migrate to higher latitudes in North America during spring and summer while males tend to migrate shorter distances and occupy mountainous regions (Cryan, 2003), and small colonies of adult females and offspring are believed to form in summer (Parsons et al., 1986; Mattson et al., 1996; Betts, 1998). Almost nothing is known about male group sizes (but they are presumed to be solitary) or survival rates. However, *L. noctivagans* appears to be an ideal species to include in comparative studies to elucidate the effects of host ecology on pathogen virulence and test Boots and Sasaki's (1999) theoretical work. Virulence factors and life history traits should also be compared to *P. subflavus*, which has recently been described as a migratory species (Fraser et al., 2012) and whose RABV variant also causes a disproportionately large proportion of human RABV cases in the USA.

- Future directions for research to address the role of host ecological strategies in driving pathogen virulence.

While some theoretical models are more advanced than field data in predicting virulence, spatial mechanistic meta-population models using parameters estimated for bats and their infections are required to determine whether highly mobile hosts can select for virulent infections. The effects of torpor, or other host factors affecting immune response, on virulence have yet to be explored, even theoretically. However, to determine the importance of host ecological strategies in pathogen virulence, experimental infection studies are required (e.g. Halpin et al., 2011); measurement of pathogen virulence (e.g. mortality or morbidity rates in hosts) is required using isolates of pathogens from migratory and non-migratory species and from those using torpor. This could be achieved using multiple isolates that are representative of the diversity bat RABV reservoirs. Ideally, results from one system (e.g. lyssaviruses) should be confirmed in others, such as the CoVs and AstVs that have been detected in bats. Studies to address the theoretical findings of Gandon (2004) will need to use infections of a common host and experimentally compare morbidity and mortality in this and other less common bat species.

Pathogen Ecology as a Driver of Bat Disease Dynamics

It is possible that the dramatic differences in migration, coloniality and torpor among bat hosts may lead to the evolution of different pathogen adaptation strategies. The differences in pathogen infection strategies (e.g. mode of transmission and virulence) and pathogen variants associated with particular hosts may drive disease dynamics beyond differences in the ecology of hosts themselves. Hampson et al. (2009) found that no matter what density of dogs was involved in an outbreak, canine RABV epidemics always had an R_0 (which is the mean number of infections caused by an infected individual in a susceptible population, Keeling and Rohani, 2008) of <2. Therefore, one might speculate that pathogen ecology drove RABV dynamics in dogs as much as host demography. Most emerging infections from bats are RNA viruses (Leroy et al., 2005; Li et al., 2005; Calisher et al., 2006; Towner et al., 2007). RNA viruses have a high capacity for mutation compared with DNA viruses due especially to the low proofreading ability of the RNA-dependent RNA polymerase that controls replication (but see Firth et al., 2010). Therefore, mutations can appear and be fixed in a short period of time relative to the host's evolutionary time frame (Moya et al., 2004). Variation in virulence, infectivity and other important processes can therefore potentially be altered within a relatively short evolutionary timescale and change the infection dynamics. However, the pathogen's ecology must also balance factors that affect transmission, such as virulence, epidemiological ‘burn-out’ and mechanisms for persistence in complex host populations (e.g. Pulliam et al., 2011).

There is little empirical evidence for differences in virulence among viruses of the same genus in their bat hosts, although the phenomenon of differing virulence among virus variants classified taxonomically as a single species is nearly universal. Studies on LBV by Markotter et al. (2009) demonstrated variable virulence among isolates when mice were challenged with different LBV isolates, as did studies with silver-haired bat RABV (Dietzschold et al., 2000). Evidence of differing virulence in humans exists for EBOV, with Reston and Cote d’Ivoire ebolaviruses not known to have caused fatal human infections (for review, see Morikawa et al., 2007), whereas other EBOV viruses and MARV have case fatality rates up to 90%. However, the virulence of these viruses in bats (the putative reservoirs) is less well described, but may be low (Swanepoel et al., 1996; Leroy et al., 2005; Towner et al., 2007). Similarly, experimental studies of henipaviruses in bat hosts have shown that in laboratory situations these viruses do not exhibit high virulence in their putative reservoir hosts (Middleton et al., 2007; Williamson et al., 1998, Williamson et al., 2000; Halpin et al., 2011) in contrast to their impacts on other species.

Maintenance strategies used by RABV and CoV, for example, clearly differ. While both have spilled over into humans and caused fatal infections (SARS-CoV perhaps through an intermediate host), they exhibit strong differences in ecological strategies. Both CoV and RABV have been detected in *E. fuscus* (Shankar et al., 2004, 2005; Dominguez et al., 2007); however, RABV infection has a low detectable prevalence, but high fitness impact on bat hosts when cerebral infection occurs, whereas CoV appears to have high infection prevalence, but a low fitness impact on bats (Drexler et al., 2011; Osborne et al., 2011). These groups of viruses also differ in modes of transmission, but both are likely transmitted through very close or direct contact of hosts. To understand infection emergence, it is necessary to understand the importance of how traits may differ between infections, with resultant differences in disease dynamics; these might then be predictable based on host and infection ecology. However, little is known about the transmission and virulence characteristics of most bat infections, and clearly, this is an important area for future study.

- Future directions for research to address how pathogen ecology drives infection dynamics.

Large-scale analyses of epidemics, such as for dog rabies (Hampson et al. (2009), will be impossible until large and comparative data sets exist for bat infections. There are few estimates of R_0 in the literature for any wildlife infection; however, Drexler et al. (2011) have shown how multiple infections can be monitored at the population level for bats and serological data can be used to estimate parameters such as force of infection (Hayman et al., 2012a). Other than steps proposed earlier to fill these data gaps that exist for almost all bat systems, it is likely necessary that further studies of within-host dynamics, and therefore experimental studies, are necessary to inform how pathogen ecology affects infection dynamics. Experimentally measuring pathogen shedding and transmission of infections with different ecological strategies in a single host may determine whether those with higher virulence (e.g. mortality or morbidity rates in hosts) have higher shedding, whereas those with lower virulence have lower shedding, but less fitness cost. These data can thus inform interpretation of field data and be used in mechanistic models.

Methods to Integrate Data and Compare the Epidemiology of Infections in Bats

We propose that multidisciplinary approaches are required to address bat ecology and disease dynamics. Detailed integrative modelling of disease dynamics in bats requires data and input from ecologists, bat biologists, pathogen experts, virologists, molecular biologists and epidemiological modellers. Integrating all these different disciplines, often through the modelling studies, is a substantial effort, often requiring a high level of technical expertise. The insights into infectious disease dynamics that can be derived from integrative modelling studies can, however, be profound, and there is a real need for more of such efforts to be undertaken. Mechanistic models also have the advantage of suggesting critical data gaps. Integrative models, such as SEIR models (Box 1), can be used to test different hypotheses, based on the relative sensitivities of parameters in the models (e.g. Buhnerkempe et al., 2011; George et al., 2011). These models can be used to predict how the impact of host and infection characteristics varies among sexes, species and diseases. In addition, quantification of the impact of other ecological characteristics can be assessed (George et al., 2011), such as bat reproduction and survival (O’Shea et al., 2004, 2010, 2011a,b), or differences in seasonal prevalence among pathogen variants. If pathogen dynamics can be significantly explained by host ecological strategies despite differences between the pathogens, it will suggest that the dynamics of emerging diseases in bats might be generally predictable from ecological characteristics.

Cyclical changes in contact and transmission rates have been suggested as the causative mechanism of temporal patterns for a variety of diseases and have been investigated using modelling approaches (Childs et al., 2000; Begon et al., 2003, 2009). Models of bat infection dynamics (for examples, see Box 1) should, therefore, integrate hypotheses regarding seasonal behaviours, including migration, coloniality and torpor use, in order to determine the effects of seasonality on disease dynamics. This approach has helped reveal the relative importance of transmission mechanisms in a plague prairie dog system (Webb et al., 2006), environmental transmission of avian influenza (Rohani et al., 2009) and in a bat RABV system (George et al., 2011). Another set of bat RABV models has been developed, but focuses largely on more detailed immunological hypotheses (Dimitrov et al., 2007, 2008), whereas other models demonstrate how altered migration behaviour may result in declining immunity within specific colonies, which can lead to more explosive HeV epidemics (Plowright et al., 2011).

If migration and coloniality are important drivers of disease dynamics, the timing and prevalence of infection are likely highly sensitive to parameters influencing rates of contact either within (George et al., 2011) or between (Plowright et al., 2011) colonies in models. Likewise, if torpor is an important driver of disease dynamics, then timing and prevalence of infection are likely highly sensitive to parameters influencing incubation period and resistance (George et al., 2011).

- Future direction for integrative modelling of bat infection dynamics.

One of the major factors prohibiting the modelling of infection dynamics in bats is the lack of appropriate data, and steps to mitigate this deficiency are described in previous sections. We have proposed elsewhere (Restif et al., in press) that for the most parsimonious study design, allowing integration of field and experimental data into models that test different hypotheses, a model-guided fieldwork approach should be undertaken. Here, model structures based on an understanding of the biology of the host and the infectious agent are used to design field studies; this should force researchers to be explicit about what is known in the system. Simulations and sensitivity analyses can be used to test differing hypotheses and parameter sensitivities, which can help the interpretation of field and laboratory data and direct future empirical data collection.

Further studies should also take comparative approaches, using models of similar but differing systems. For example, although bat hosts of RABV and other lyssaviruses vary in some host ecological strategies, such as in the use of torpor, the question arises as to whether common traits, such as seasonal birth pulses, can be used to predict the infection dynamics across lyssavirus systems. If so, this will provide a powerful insight into the drivers of infection dynamics, so general predictions regarding infection ecology and spillover can be made.

Challenges to Understanding the Ecology of Infectious Agents and their Bat Hosts

There are substantial difficulties in performing these integrated and multidisciplinary studies in most bat–pathogen systems, including those associated with pathogen detection, time series data collection and significant variation in host ecological parameters, both within and among species. The detection of infections with low prevalence, such as those caused by some RNA viruses, can be very difficult and may require non-random sampling, very large sample sizes, specialized techniques or a combination of all three (e.g. Leroy et al., 2005; Kuzmin et al., 2008b). The detection of EBOV RNA in bats is one clear example (Leroy et al., 2005). It took decades to discover EBOV RNA in bats, despite intensive efforts and epidemiological and experimental evidence linking the infection to bats (Swanepoel et al., 1996; Monath, 1999; Pourrut et al., 2005), and to date, the viruses are yet to be isolated by cell culture from any bat. Furthermore, although bats were strongly implicated as reservoirs to SARS-like CoVs and diverse lineages of CoVs have been detected from bats globally, to date there has been no CoV isolate from bats because of the non-permissive nature of extant cell culture systems.

Time series data are requisite for tackling complexities in inter-annual variation in disease ecology, but efforts to collect them can be immense. Drexler et al. (2011) studied a maternity colony of *M. myotis* bats in the attic of a private house in a suburban neighbourhood in Germany, during 2008, 2009 and 2010. This was an elegant study; however, it was only performed on a single maternity colony and only for a short period of each year; furthermore, samples and data were only available at the population scale. Similar population-level studies have been reported for HeV and NiV in Australia and Thailand (Wacharapluesadee et al., 2010; Field et al., 2011). All three of these studies, however, captured seasonality in viral shedding. Case submission data have also been used to capture seasonality, such as for RABV submissions used by George et al. (2011); however, these data are biased (see Klug et al. 2011) and unlikely to be relevant for infections with low mortality in the host. Studies looking for individuals shedding infection have been less successful. For example, Hayman et al. (2012a) failed to detect lyssavirus RNA in oral swabs from 796

E. helvum bats sampled over four years. Despite seemingly large overall sample size, the individual sampling events were unlikely to detect low infection prevalence and were further reduced when the authors accounted for the testing process sensitivity. If detection of the infection can be difficult, determination of the mode of transmission, periods of infectiousness and rates of recovery can be even more challenging and often require experimental studies. However, experimental studies in bats, such as those with RABV and other lyssaviruses (Hughes et al., 2006; Franka et al., 2008; Kuzmin et al., 2008a; Turmelle et al., 2010b), EBOV (Swanepoel et al., 1996) and HeV (Williamson et al., 1998) can be very costly, usually involve small sample sizes and highlight the complexities of host response to infection (Franka et al., 2008; Turmelle et al., 2010b; Halpin et al., 2011). Additionally, measuring morbidity and mortality in animals experimentally infected with potentially highly pathogenic infection can raise difficult ethical questions. Although not without their own ethical issues, initially, standard mouse models may be instructive, because they are of known infection history, genetically homologous (thus reducing host heterogeneity that might obscure mechanistic trends), and there are a range of immune markers available that do not exist for bats; however, studies using mice will not address important questions on dynamics of infection in natural hosts. Further difficulties exist in testing some hypotheses, because though apparently widespread in bats, infection such as CoVs and AstV are yet to have been propagated in the laboratory.

Complications also arise when attempting to estimate vital host-related parameters. Birth rates are generally measurable, but mortality rates and the causes of mortality are particularly challenging to determine in wild populations (O'Shea et al., 2004). Generally capture–mark–recapture methods are used for estimating mortality rates; however, these studies are difficult, especially with migratory species or species with large colony sizes (Hayman et al., 2012b). Determination of the true age of wild bats is also difficult once bats reach maturity, making life table analyses intractable (life table approaches can also have analytical shortcomings; Williams et al., 2002). Hayman et al. (2012a) used tooth cementum annuli to age bats and determined that age-specific seroprevalence against LBV and estimated force of infection increased with age (Hayman et al., 2012a). However, this was a destructive process (canine teeth were used) and the sample size small.

The quantification of seasonal and sex differences in coloniality, torpor and migration is difficult because of peculiarities in the ecology and life histories of bats themselves. Typically, adequate information only exists for parts of the year (one particular season; Hayman et al., 2012a,b; Drexler et al., 2011) or one particular sex (Weller et al., 2009; Drexler et al., 2011). For example, information exists on summer colony sizes of *E. fuscus* and activity patterns of females, but not males; however, population genetic studies have shed some light on sex-biased patterns of dispersal in this species (Nadin-Davis et al., 2010; Turmelle et al., 2011). For other species, such as *E. helvum,* migration patterns are largely unknown, so information is only available from colonies that are conspicuous for parts of the year (Sorensen and Halberg, 2001; Richter and Cumming, 2006, 2008; Hayman et al., 2010, 2012a,b). Studying non-migratory colonies of some species may provide insight into the relative effects of migration and other ecological factors (Peel et al., 2012). However, group and population sizes themselves may be very difficult to determine (Kunz, 2003), and roosting dynamics may be hard to quantify. Individuals in large colonies are difficult to identify and count, and small colonies may be inconspicuous (Weller et al., 2009). Often, only roosting or foraging behaviour is known, and tracking over long distances requires more labour-intensive methods, such as tagging with wing bands or following bats fitted with radio transmitters using fixed-wing aircraft (Cryan and Diehl, 2009). Precise GPS or satellite tracking systems are currently too large for long-distance tracking of all but the largest bat species (Smith et al., 2011), although these studies have shed light on movement of *Pteropus* in the context of NiV and HeV dynamics (Epstein et al., 2009; Breed et al., 2010; Smith et al., 2011). The use of infrared video cameras has recently been utilized to monitor roost entrances and count the number of bats that emerge each night and associated host thermal energetic profiles (Hristov et al., 2008; Reichard et al., 2010a, 2010b), however, these systems are costly and in their infancy with respect to general use.

- Advances required to improve the understanding of infection ecology in bats.

Far wider use of capture–recapture techniques is required to obtain estimates of host survival and infection-related parameters. While manual capture has distinct advantages, such as allowing sampling of individuals to obtain infection-related data, it may be impractical in many instances. Thus, wider use of passive integrated responders (PIT; microchips) and readers (see O'Shea et al., 2011a,b) for marking bats and the use of telemetry will be necessary. Hayman et al. (2012b) used radio transmitters simply as a method for 'presence–absence' detection in a capture–recapture study of migratory *E. helvum*. Currently, transmitter size limits the use of telemetry in most bat species (Smith et al., 2011), and the range limits the use of PIT tags. Thus, improved micro-technologies are required to reduce the size of the transmitters without compromising battery capacity. These technologies are also valuable to locate the sites bats migrate to or hibernate in.

Monitoring colony sizes has been of concern for bat ecologists for a number of years, and new technologies are being developed to gain more accurate estimates (e.g. Hristov et al., 2010). These estimates are necessary to test specific hypotheses relating to critical community size, meta-population infection dynamics and coloniality. However, the use of such technologies is still in development, as are the computer algorithms to estimate abundance, and thus, widespread monitoring of colonies is currently very limited.

Interpretation of infection-related data, such as antibody prevalence, is problematic if the age of the animals sampled is unknown; therefore, better techniques to age bats are required. Hayman et al. (2012a,b) used tooth cementum annuli, but clearly the destructive sampling is not appropriate in the majority of situations. Thus, in the absence of data from individually marked bats, non-destructive sampling from teeth might be a productive area for research (e.g. Plowright et al., 2008a).

For better interpretation of infection-related data, immunological tools such as bat-specific reagents, for example, are necessary to be able to understand bat immune responses to infection (whether from field or experimental data). Permissive bat cell lines (e.g. Crameri et al., 2009) may be necessary for isolation of viruses, and experimental studies are necessary to understand the within-host dynamics and pathogenesis of infections in bats and modes of transmission. Highly variable serological responses have been observed among individual bats in experimental studies, meaning interpretation of results is not always straightforward (Turmelle et al., 2010b, Halpin et al., 2011). These experimental studies must be designed to better understand empirical field data (e.g. Sulkin et al., 1960). In particular, baseline estimates of antibody responses from naive bats (e.g. captive bred) are necessary to better interpret serological results from field studies (see Williamson et al., 1998, 2000; Turmelle et al., 2010b). Regarding bat immune responses, typically adaptive responses are measured because tools are available to measure antibody responses. However, substantial advancements in the understanding of bat infection ecology will be made once innate immune responses are understood. This is particularly important for infections such as CoV, filoviruses and henipaviruses, which apparently do not cause overt disease in bats. Cytokine expression profiles, for example, will provide a much deeper understanding of bat immune responses following infection (for an example of methods, see Sadeghi et al., 2011).

Lastly, while suitable statistical and modelling methods exist already, and generally a lack of data is the issue, better use of specific approaches are required. Specifically, increased use of mechanistic models to integrate data from different sources is needed to understand the driver of infection dynamics, and sensitivity analyses should be used once these models have been developed (Restif et al., in press). Consideration of meta-population dynamics and multispecies/pathogen models may be necessary to understand some systems; however, these models suffer from even greater issues relating to parameterization than single species–single host models. For statistical analysis, use of Bayesian frameworks should be considered when some prior data or knowledge exists and/or sample sizes are small. Bayesian analysis is often better able to estimate parameters from small data sets than frequentist methods, plus have the flexibility to incorporate prior knowledge into parameter estimates (Colchero and Clark, 2012). Both these characteristics may be appropriate for studies of bats when little information is known on a specific species and samples sizes are generally small.

Understanding Anthropogenic Changes

Although human–bat interactions are difficult to quantify, it appears that such interactions are increasing and occurring in numerous ways. Increasingly, humans encroach into bat habitats, and bats are utilizing artificial structures as roosts. Examples of bats utilizing artificial structures include *M. myotis* (infected with AstV, CoV and AdV) roosting in homes in Europe (Drexler et al., 2011) and RABV-infected *T. brasiliensis* and *E. fuscus* bats in bridges and houses in the Americas (Shankar et al., 2005; Turmelle et al., 2010a; O'Shea et al., 2011a,b). CoV-infected *E. fuscus* have been detected in homes in the United States also (Osborne et al., 2011). Several MARV spillover cases have been linked epidemiologically to cave activities such as tourism and mining (for review, see Towner et al., 2009) and SARS-CoV is believed to have emerged because of the use of bats as food in live 'wet' markets in China (Woo et al., 2006). Nipah virus outbreaks have been linked to palm sap collection in Bangladesh and intensification of pig farming, alongside mango (*Mangifera indica*) production in Malaysia (Luby et al., 2006; Nahar et al., 2011; Khan et al., 2011; Pulliam et al., 2011). Hendra virus outbreaks have been suggested to be attributable to increased urban habituation of bats in Australia (Plowright et al., 2011). Recent surveys suggest that the numbers of harvested *Pteropus* and *Eidolon* bats (old world fruit bats) in Asia and Africa can be very high (and likely unsustainable, see Kamins et al., 2011; Struebig et al., 2007; Epstein et al., 2009). The harvested numbers estimated from single regions within nations suggest that human–bat interactions are occurring on an enormous scale throughout both continents. Choisy and Rohani (2006) used theoretical models to predict that in many systems harvesting may increase the prevalence of infection and size of epidemic peaks in populations of harvested animals, because density-dependent recruitment (through increased survival of young) increases the susceptible pool and hence the size of the epidemic peak in populations. These hunting figures, coupled with the increasing use of human-built shelters by bats, mean that the dynamics at the human–bat population interface are a neglected area of research. We believe that this gap must be addressed if we are to understand spillover risk.

Additionally, attitudes to bats and bat conservation must be understood to prevent further spillover (Olival et al., 2012). It is encouraging, however, that recent examples appear to demonstrate that if addressed appropriately, mitigation strategies can be implemented (Nahar et al., 2011; Stone, 2011). However, when considering zoonotic infections, bat infection spillover events to humans are typically so infrequent that human effects on spillover dynamics may be difficult to detect. For example, did reduced encroachment and intermediate host contact in Malaysia, because of laws preventing fruit farming in pig farming areas, prevent further NiV outbreaks in Malaysia? In Bangladesh, where NiV outbreaks are almost annual, it may be easier to quantify if preventative measures are successful (Nahar et al., 2011; Stone, 2011). However, neither spillover prevention method may be affecting bat infection dynamics themselves.

- Advances required to improve the understanding of anthropological effects on infection ecology in bats.

Only once well-parameterized models of systems are developed, can perturbations, such as through harvesting or restricting bat movement, be simulated to attempt to predict alterations in infection dynamics. Empirical evidence can be more difficult to produce, in particular because control studies may be difficult to perform, particularly in migratory species, but attempts should be made. Opportunistic (but not poorly designed) studies may be necessary. For example, some agencies are using destructive control methods to eliminate species such as vampire bats to prevent RABV spillover infections in cattle and people. Monitoring population and infection dynamics in both these and untouched populations could provide evidence of increased intensity of epidemics in the local populations, as predicted by Choisy and Rohani (2006). Where culling or hunting occurs in Africa and Asia (Struebig et al., 2007; Epstein et al., 2009; Kamins et al., 2011), studying infection dynamics may be illuminating. Clearly in all cases, studies are necessary for both conservation and infection spillover purposes.

Summary

Understanding disease dynamics in wildlife populations, specifically within populations of bats, is important if informed policy and mitigation strategies are to be taken. The increase in human and bat contact is inevitable; however, there is potential to manage how these interactions occur (Nahar et al., 2011; Stone, 2011). We propose a combination of field and laboratory studies that can be used to create well-validated data-driven mechanistic models to elucidate the aspects of bat ecology that are most critical to the dynamics of emerging bat viruses. This trait-based predictive approach will be particularly important when population surveillance data are unavailable, and it is unclear which aspects of host ecology may be most important in driving potential disease emergence.

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